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GLAUCOMA AND ITS TREATMENT

G. L. JOHNSON

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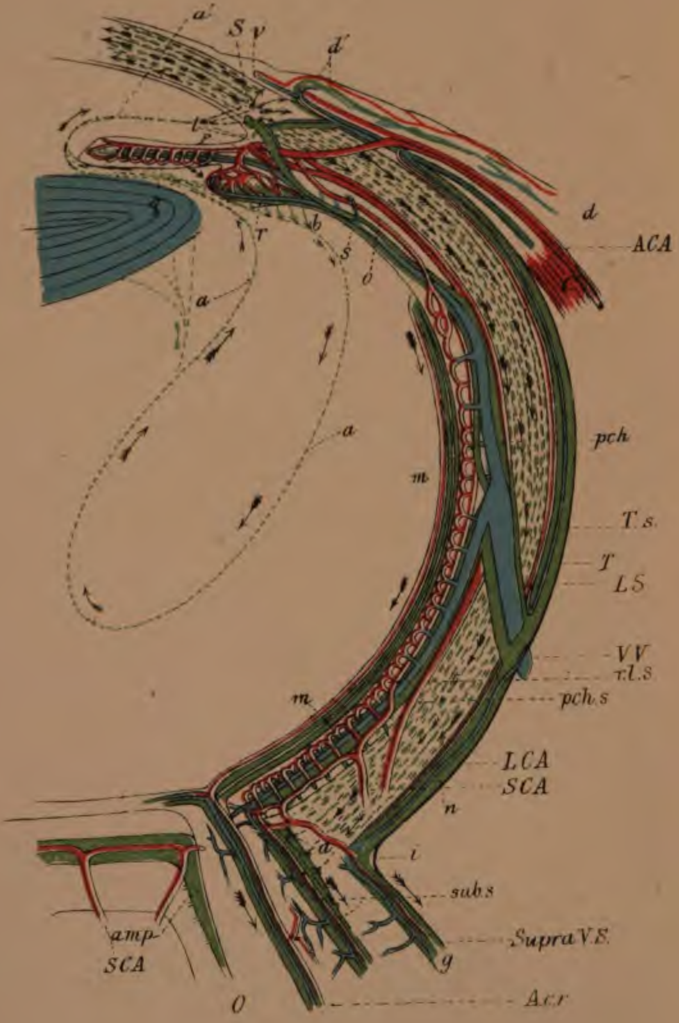
A NEW METHOD
OF TREATING
CHRONIC GLAUCOMA.

DIAGRAM OF THE LYMPHATIC & VASCULAR SYSTEMS OF THE EYE.



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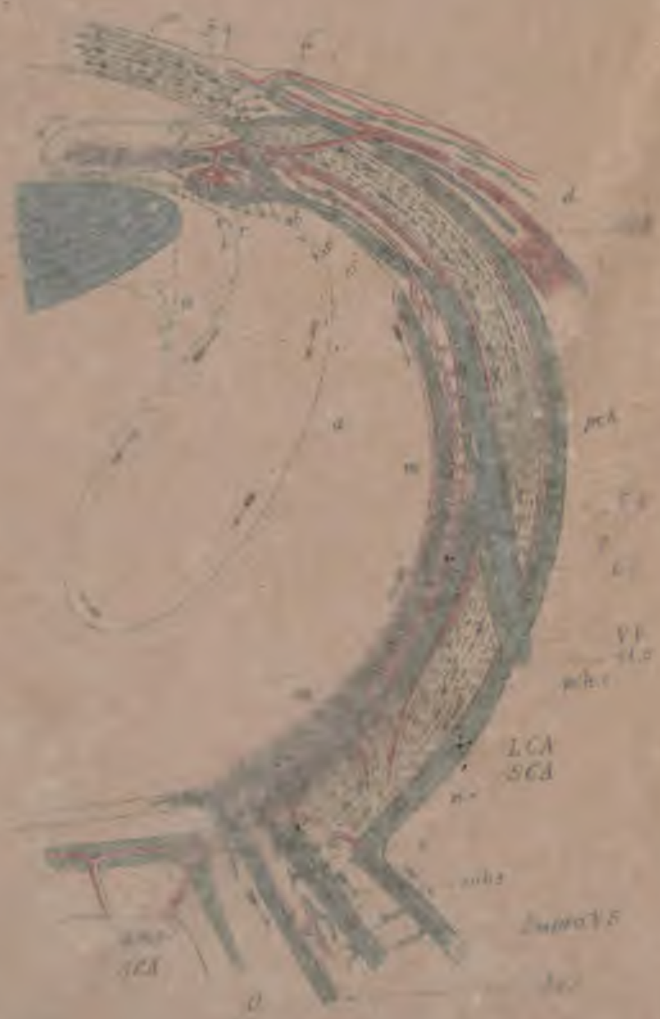

A NEW METHOD

OF TREATING

CHRONIC GLAUCOMA

BASED ON

RECENT RESEARCHES INTO ITS PATHOLOGY.

BY

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WITH ILLUSTRATIONS.

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PREFACE.

THE beginning of my studies of this subject, was a thesis for the M.B. degree read at Cambridge in 1882, which had the good fortune to be specially commended by the Regius Professor of Physic.

Dr. Paget advised me to publish my thesis, and this book is the result of his suggestion. While it has been necessary to write more fully on several points than was possible in a thesis, I have endeavoured to be concise, and have only added what was necessary to elucidate the subject as a whole, and to bring it up to the level of our present knowledge.

My best thanks are due to Dr. Albert Gresswell, for revising the proofs; to Mr. Power, Mr. Rouse, Mr. Cowell, and the other surgeons at the Westminster and Moorfields' Eye Hospitals, for giving me so many opportunities of observation and research. Lastly I am deeply indebted to Dr. Brailey, for placing the magnificent collection of pathological slides at Moorfields at my disposal.

London. June, 1884

DESCRIPTION OF FRONTISPIECE.

Diagram of the Lymphatic and Vascular Systems of the Eye.

The vascular schema is copied from Leber's diagram in Stricker's Handbook. The lymphatic schema I have constructed partly from my own observations, partly from those of others.

The arteries are coloured red, the veins and lens blue, and the lymphatics green.

The course which the lymph takes from the Iris and Ciliary processes to Schlemm's canal and along the Cornea and Sclerotic is indicated by dotted lines.

L.C.A. Long ciliary artery.
S.C.A. Short ciliary artery.
A.C.A. Anterior ciliary artery.
A.c.r. Arteria centralis retinæ.
VV. Vena vorticosa.
C. Rectus muscle.
O. Optic nerve.
S. Schlemm's canal.
T. Tenon's capsule.
T.s. Tenon's lymph space.
pch.s. Perichoroidal lymph space.
Sub.s. Subarachnoid lymph space.
Supra. V.S. Supravaginal lymph space.
r.l.s. Retinal lymph space.
L.S. Lymph space around the vena vorticosa.
amp. Ampulla of De Wecker, a dilatation of the subarachnoid (subvaginal) space, situated between the pial and arachnoid sheaths of the optic nerve.
a. Track of lymph current through the media of the eye.
b. Lymph streams passing into the vitreous from the ciliary processes.
d. Conjunctiva.
d′. Its bloodvessels.
g. Vessels of the iris.

"Let a man use his own experience as best he can for the present, but let him not, upon the strength of it, rebuke the experience of all past times, and dictate to the experience of all future; for if he lives long enough, nothing is more likely than that he may find himself fallen under his own reproof and inconveniently confronted by his own maxims."

Dr. Latham.
"*Clinical Lectures.*"

A NEW METHOD OF TREATING CHRONIC GLAUCOMA.

PART I.

THE object of these pages is to endeavour by a process of inductive reasoning to establish the truth of the following proposition, and to suggest a remedy for chronic glaucoma which is not open to the objections inevitable to iridectomy. The proposition I wish to establish is the following:—

"That the ordinary method of treatment for glaucoma by iridectomy though highly successful in acute forms of the disease, is nevertheless both uncertain and unsatisfactory in the chronic condition of glaucoma."

I believe I am not overstepping the mark when I say that there is no disease in the whole history of ophthalmology which has been so diligently enquired into, and at the same time so little understood, as glaucoma. From the very earliest times, surgeons have taken refuge in theories to mask their ignorance of this disease. Remarkably enough, until recent times the degree of ocular tension had never attracted much notice nor was its value appreciated, and in consequence of this, as we have abundant proof to show, glaucoma and cataract could not be distinguished. The later Greeks divided the disease into two classes; to those cases which were incurable they gave the name γλαυκώματα, while those which were capable of some improvement were called ὑπόχυματα.

Cataract and glaucoma were both considered diseases of the lens, and were deemed merely different stages of the same disease. Even so late as 1557 Oribasius confuses the two, stating that glaucoma is a conversion of the vitreous humour into a blue substance, and only occasionally curable.[*] Such views had but little advanced even in Mackenzie's time, for he gives elaborate directions by which the student might distinguish between glaucoma, amaurosis, and cataract, nor was it until Helmholtz discovered the properties of the ophthalmoscope in 1851, that surgeons were able to recognise the true features of the disease. With such vague notions of the real nature of the disease it cannot be wondered at, that the treatment was equally unsatisfactory. Mercury, antimony, blisters, bleeding, and even the removal of the lens, were in turn recommended. Mackenzie so late as 1835 writes, "On the presumption that glaucoma originates in an inflammatory affection of the hyaloid membrane, bleeding and purging have been employed for its cure, and occasionally this practice has been attended with benefit." He then goes on to say, "when glaucoma has commenced in one eye it generally extends to the other: in its fully formed stage glaucoma is absolutely incurable, but remedies may occasionally arrest the progress of the disease." It might literally be said of glaucoma that it was a disease, in which the patient saw nothing and the physician saw nothing. Things remained in this state until 1857, when Von Graefe by removing a portion of the iris in a case of glaucoma, accidently made that brilliant discovery which has made his name so justly famous. "It is hardly too much to say that before Von Graefe's time every eye attacked by true glaucoma became blind, unless death overtook its possessor, before the disease had had time to run its course."* But although the astonishing results which frequently followed iridectomy in patients suffering from acute glaucoma, led to its universal adoption; nevertheless, a large number of cases presented themselves, in which iridectomy completely failed to give the slightest relief, and the eye in spite of every care lapsed slowly into a state of amaurosis. Hence it is, that surgeons unsatisfied with iridectomy, strove to solve the problem by the use of other remedies. Sclerotomy, intraocular myotomy, trephining the sclerotic, division of the ciliary nerves, or of the sheath of the optic nerve, and in medicines mercury, ergot of rye, opium, eserine, jaborandi and pilocarpine, are only a few of the operations and drugs that have been held out as specifics. That all these remedies have been of value in certain cases of glaucoma is undoubted, indeed I am inclined to think the reason of their successive abandonment, lies in the ignorance of the true nature and pathology of the disease and their consequent application to unsuitable cases.* Why is iridectomy so wonderfully successful in some cases of glaucoma and so signal a failure in others? Why does eserine relieve tension in some cases and yet intensify an attack of glaucoma in others? To answer these questions aright, we must examine the course and nature of the pathological process, and we believe we can demonstrate not only wherein Von Graefe's operation lacks success, but on purely pathological grounds we can suggest a remedy for those very cases which iridectomy fails to benefit.

---

[*] Glaucoma et suffusionem veteris unum eundemque morbum esse existimarunt; posteriores verò glaucomata humoris glacialis qui ex proprio colore in glaucum convertatur et mutetur morbum esse putaverunt: suffusionem verò esse effusionem humorum inter uveam et crystalloidem tunicam concrescentium; cæterum glaucomata omnia curationem non recipiunt suffusiones verò recipiunt sed non omnes. *Oribasii Synopseos*, lib. vii, chap. 47.

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* Priestley Smith. *Jacksonian Essay*, 1879.

I define glaucoma to be the sum of the pathological conditions dependent on increased tension of the eyeball, from whatever cause arising.

All varieties of glaucoma whether primary, or secondary (*i.e.* consequent on some previous disease or injury), resolve themselves under two main symptomatic heads:—1. That in which the symptoms are comparatively rapid in their onset, with intense pain, congestion of the eyeball, and turbidity of the media, often accompanied by great swelling and inflammation of the iris and ciliary processes, and active arterial hyperæmia. This I will call acute glaucoma, it includes the fulminating form of Von Graefe, and serous iritis. 2. That form in which the symptoms are characterized by a remarkable insidiousness and chronicity, by the absence of a sudden onset, little pain, and very slight turbidity of the media; and as a general rule marked excavation of the optic nerve. Occasionally there may be exacerbations of an irritative or quasi-inflammatory type. This I will call chronic glaucoma, and it includes the subacute form of Von Graefe. Traumatic, hæmorrhagic, and absolute glaucoma do not for the requirements of this essay, need a separate classification.

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* Vide Appendix, Note 1.

The glaucomatous condition is directly due to a disturbance between the normal relations of secretion and excretion of lymph within the globe. Whenever the secretion is in excess of that removed by the vessels, an increase of tension results, and glaucoma is established.

I know of only three ways by which this result can be brought about:—

(1). By vasomotor nerve influence, by which the calibre of the vessels is altered, and the rate of secretion increased.

(2). By acute inflammation or by passive hyperæmia of the ciliary body and tunica vasculosa, which cause an increased secretion, and in some cases a retarded excretion.

(3). Lastly, by contraction or occlusion of the lymphatic waste channels, whereby the outflow of lymph is retarded. This is by far the commonest and often the only cause of glaucoma, but the two former ways take a share in a large number of cases.

Let us examine the subject a little more closely.

The lymph which nourishes the tissues of the eye

FIG. I.

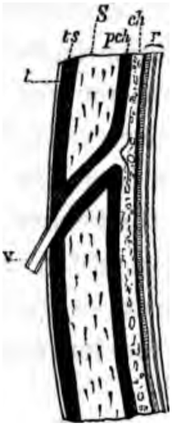

Diagram showing the communications between Tenon's space and the perichoroidal lymph channel. It represents a section taken through the tunics of the globe at the point where one of the great veins emerges. (After Schwalbe)

r. Retina. *ch.* Choroid. S. Sclerotic. *t.* Tenon's capsule. *pch.* Perichoroidal space. *ts.* Tenon's space. *v.* Vena vorticosa.

is secreted by the ciliary processes, and probably to a small extent by the iris, but whether the choroid takes any share in this under normal conditions is a matter of considerable doubt. The amount secreted by the iris must be very small, and is rather inferred from its resemblance in structure to the ciliary processes than capable of actual proof. The share taken by the choroid in the process, is still more hypothetical.

The fluid secreted by the ciliary processes passes at once throughout the minute lymph clefts of the vitreous.* After permeating its entire structure, the lymph returns through the suspensory ligament, between the lens and ciliary processes into the posterior aqueous chamber; and thence partly between the iris and the lens capsule, and partly through the base of the iris into the anterior chamber. A small quantity passes directly from the ciliary processes into the posterior aqueous chamber behind the iris, where it joins the main current. (See Frontispiece). Under normal conditions, the iris rests against the lens so closely as to form a kind of valve, which only allows of a current in a forward direction, any pressure in the anterior chamber only shutting the valve the closer. The lymph in the anterior chamber percolates by osmosis through a number of fine meshes and minute fissures which exist around the angle of the anterior chamber near to the point of constriction of Descemet's membrane, known as the spaces of Fontana. Here we can trace three currents; one passes into the lymph spaces of the cornea to nourish that part; a second percolates through similar lymph spaces in the sclerotic; while the main stream discharges itself into the canal of Schlemm whence it passes into the anterior ciliary veins. The manner in which the canal of Schlemm is connected with the veins in its vicinity is still unknown. (Vide Frontispiece). In all probability certain valvular arrangements exist, which prevent the passage of venous blood into the canal of Schlemm, under the normal conditions of pressure. Were the lymph of the anterior chamber to empty itself directly into the lymphatics, so rapid an outflow would occur that the sluggish transudation of fresh fluid into the chamber could not be possibly compensated for, and the cornea would collapse. This, however, is prevented by the intervention of the canal of Schlemm, which does duty as a regulator between the two. In the smaller veins the pressure is always higher than in the corresponding lymphatics; moreover the trabecular meshes offer resistance to the outflowing lymph; by this means the balance of pressure in the two sets of vessels, is maintained. Injections into the anterior chamber can be readily made to pass into the anterior ciliary veins in the above manner. The anterior chamber does not communicate *directly*, either with the perichoroidal space,

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* See note 2 in the appendix.

Tenon's space, or with the lymphatics of the conjunctiva; but it does so *indirectly* through the communications of the spaces of Fontana with the canalicular system of the cornea. This canalicular system deserves a moment's notice. The cornea, as everyone knows, consists of from 60 to 70 fibrous layers, each layer being separated from the next by a homogeneous matrix. In the latter lie the cell spaces. These are many-branched flat cavities or lacunæ, freely anastomosing with similar lacunæ on all sides by narrow irregular shaped canaliculi. They are merely excavations in the matrix having no lining membrane, each lacuna being almost entirely filled up by a flat branched cell, the corneal corpuscle. Between the corpuscle and the lacuna wall there is just room for the passage of lymph and a single layer of migratory cells, so that the lymph can pass in this way from one side of the cornea to the other, or even right round the eyeball; for, as we have previously stated, this canalicular system is continued throughout the sclerotic. Moreover as there is a network of these lacunæ between every two layers of corneal fibres, the total amount of lymph circulating through the cornea and sclerotic is very great.*

---

* I wish to lay particular stress on this point because it largely affects my theory. The lymphatic system of the cornea and sclerotic is minutely described in a recent and very able course of lectures by Ranvier "*Leçons sur les Terminations Nerveuse Cornée.*"

All those parts of the globe situated behind the ciliary body, outside the vitreous chamber, discharge their lymph in one of two ways. That from the retina by a tract within the optic nerve, that from the choroid sclerotic and cornea, escapes along the lymph spaces round the venæ vorticosæ, (fig. 1) and then passes directly into a lacuniform space (the perichoroidal space) situated between the sclerotic and choroid along their whole extent from the ciliary body to the optic nerve. At the optic nerve the perichoroidal space communicates directly with the subarachnoid space, a well marked lymph channel, lying between the two sheaths of the optic nerve, and opening directly into the subarachnoid cavity of the brain.

The walls of the perichoroidal space are held together by a trabecular tissue, composed of numerous lamellæ of elastic fibres and branched connective tissue corpuscles. These lamellæ are covered with an endothelium, like that of ordinary lymph canals, and are separated from one another by lymph spaces. On the outside of the sclerotic, is another similar lymph channel (Tenon's space). The blood of the choroid is removed by four large veins (the venæ vorticosæ) which piercing the sclerotic open directly into the ophthalmic vein. A perivascular lymphatic sheath communicating with both the above spaces, surrounds each vein in its passage through the sclerotic, (see fig. 1).

At the posterior pole of the eye Tenon's capsule* forms a covering for the external sheath of the optic nerve, between these two therefore, is the continuation of Tenon's space (supravaginal space), which we have already described as opening directly through the canalis opticus into the arachnoid cavity of the brain.

The lymphatics of the retina are perivascular canals, similar to those around the vessels of the brain and spinal cord. The veins and capillaries are completely invested by these sheaths, but the arteries only partially so. A minute serous cavity also exists between the membrana limitans interna and the nerve fibre layer, which communicates with these channels, so that the lymph has a ready exit through the lamina cribrosa, and this it does the more readily, owing to its communication with the above mentioned subarachnoid and perichoroidal spaces.

To sum up therefore; any fluid secreted by the ciliary processes or iris, after permeating the vitreous and aqueous humours passes into the anterior ciliary veins by way of the intertrabecular spaces of Fontana and Schlemm's canal; while any fluid in the choroid, retina, sclerotic or cornea, will readily find its way into the arachnoid or subarachnoid cavities of the brain. Now when we consider the immense number of lymph channels, running in parallel planes throughout the cornea and sclerotic, and the ready means of exit along the perichoroidal, subarachnoid and Tenonian channels, which are I believe always permanent means of exit, we cannot fail to see how complete and efficient a system of drainage we have here capable of being made use of, were the approaches to Schlemm's canal obstructed.

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* See note 3 in appendix.

We have hitherto repeatedly spoken of the fluid secreted by the ciliary processes and iris as lymph, and we have done this in conformity with nearly every writer on the subject. But is it lymph? I think we may regard the anterior chamber as a lymph sac, lined throughout with a delicate tesselated layer of cells (for the cells of Descemet's membrane can be traced uninterruptedly round the angle over the whole of the anterior surface of the iris, right up to the pupillary margin), and we know from experiments that the amount of fluid secreted into the anterior chamber is in a measure dependent on the blood pressure in the neighbouring arteries. Prima facie the case appears straight-forward enough, and we should seem scarcely justified in raising the question. But if we accept the traditional doctrine, we are confronted at the outset, with the difficulty that the composition of lymph and aqueous are quite different, indeed analysis shows that aqueous agrees far more closely with vitreous than it does with lymph, as the following tables show.

Aqueous (man).
Sp. Gravity 1006

Water	986·87
Proteids	**1·22**
Traces of urea, Extractives, etc.	4·21
Sodium chloride	6·89
Other mineral matters	0·81
Fat	**0·00**
Total	1000·

Vitreous (man) Lohmeyer.

Water	986·400
Membranes	·210
Mucin	1·360
Fats	·016
Extractives	3·206
Sodium chloride	7·757
Other minerals	1·051
Total	1000·

Lymph (human) Lang.

Water	943·6
Albumin	**21·2**
Fibrin	1·6
Fat	**24·8**
Extractives	1·6
Salts (chiefly NaCl)	7·2
Total	1000·

Liquor Sanguinis (man).
Schmidt and Lehmann.

Water	902·90
Proteids (chiefly serum albumin)	78·84
Fibrin	4·05
Fat	1·72
Extractives	3·94
Salts (chiefly NaCl)	8·55
Total	1000·

Analysis of Pleural and Peritoneal fluids during life from man by Paracentesis (Scherer).

	Pleura	Peritoneum
Water	935·52	953·00
Albumin	49·77	34·58
Fibrin	0·62	0·32
Extractives	5·60	4·28
Salts	7·93	7·22
Total	1000·	1000·

In comparing the vitreous with the aqueous, we must of course bear in mind that the former consists in greater part of primitive mesoblastic elements, and resembles a preformed tissue rather than a secreted fluid. Nevertheless they resemble each other far more closely than do lymph and aqueous. When we examine the above analysis of blood lymph (which is taken from an average of a large number of specimens) we find the albumin and fat largely in excess of that found in the aqueous humour. This is only what we should expect when we consider the enormous amount of metabolic activity which takes place everywhere in muscular contraction, and for which this richly fat and proteid laden fluid is admirably suited for restoring the expended energy. Thus in a thousand parts of lymph we find 21·2 parts of albumin and 24·8 parts of fat, whereas the fat is entirely absent in aqueous, indeed all the proteids together only amount to 1·2 per 1000. How can this difference be explained? It is obvious, that a mere filtration of lymph through the ciliary processes, and ligamentum pectinatum, could not possibly give rise to so great an alteration in the constituents of the two liquids. The amount of proteid material in pleural and peritoneal fluids, is intermediate between that contained in lymph and liquor sanguinis, and certainly there is a closer connection between the composition of these fluids than between them and aqueous.

Now although there is nothing within the eye itself akin to gland tissue, yet there are certain cells in connection with the tunica vasculosa and retina, which do perform distinct metabolic functions. To give an example, Kühne in his elaborate investigation into the nature of the visual purple,* has established beyond doubt the fact, that the amount of visual purple (rhodopsin) contained in the rods, is in direct proportion to the activity of the hexagonal pigment cells, against which the ends of the rods rest. Indeed, the way in which these cells elaborate rhodopsin, is precisely analogous to the way in which the cells of a gland produce mucin at the expense of their own protoplasm.

Now these cells are continuous both histologically and morphologically, with the cells which line the ciliary processes and the posterior surface of the iris, forming its uveal layer.† This layer is very conspicuous, and is distinctly traceable beneath the pars ciliaris retinæ, ensheathing each ciliary process with so thick a fold as to form almost its entire structure. From these processes it is continued along the posterior surface of the iris, as far as its pupillary margin.

What purpose do these cells serve in this part of the eye, beyond the mere mechanical one of absorbing the light and preventing reflection, by reason of their dense pigment ? If in one part of the eye they take on themselves a distinct metabolic function, do similar cells in another part of the eye necessarily lose this property altogether? Now Leber and others have established beyond controversy, that the aqueous (?) fluid is secreted into the eye from the ciliary processes and posterior surface of the iris ; and more over, Deutschmann at the suggestion of Professor Leber, has arrived at the same conclusion, by what may be termed a negative experiment. By using great precautions, he removed the entire iris and ciliary processes from a rabbit's eye, without causing the slightest inflammation. Soon afterwards the eyes were observed to become soft, and not only was the aqueous humour not renewed but even the vitreous disappeared through deprivation of its infiltrating nutriment.

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* Kühne. *Zur Photochemie der Netzhaut*, and *Ueber den Sehpurpur*, Heidelberg, 1877. Also, *Untersuchungen aus dem Physiologischen Institute der Universität Heidelberg*, Vol. I., Part I, II, III, and IV.

† Klein. *Atlas of Histology*, p. 360.

Taking it for granted that the intraocular fluid is secreted by the iris and ciliary processes, I think then, we must all arrive at the conclusion that these cells are mainly, though possibly as we shall shortly show, not *entirely*, responsible for the elaboration of the intraocular fluid.

Considered in this light, it is highly probable that the remarkably plicate condition of the ciliary processes serves a double purpose. For it not only affords a large secreting surface for the rapid supply of fluid, but also by distributing the cells which take on this selective action, over a wide area, allows room for an immense number of them, and thus the work falls, so to speak, insensibly on each one, while the space occupied is economised, as in the familiar examples of the glomeruli of sweat-glands and the kidney.

There is no doubt that hitherto, surgeons have attributed far too many diseases of the eye to inflammation. One cannot fail to be struck with the frequency one meets with the termination—*itis* in connection with diseases of the ocular tunics. Must diseases of the choroid invariably be due to inflammatory changes? Are all changes in the retina, excepting mechanical injuries and detachment, to be attributed, (at least primarily), to inflammation? If we feed a child on an unsuitable diet, we shall soon establish a disease, and if the nature of the secretory cells of the tunica vasculosa becomes altered, the fluid secreted will be chemically altered as well. Now I think I am right in arguing, that upon this fluid which supplies nourishment to the eye, the health of the tunics directly depend, and that many of the diseases which we now ascribe to inflammation, are really due to failure on the part of the cells of the tunica vasculosa to perform their proper functions. I have often pointed out the relationship which exists between degeneration of these cells and many forms of cataract. I am of opinion that the integrity and healthy condition of these cells are of the highest importance to the health of the tunics and especially to the vitreous and lens, and that cataract is not the only disease which can be laid to the charge of their perverted action. Do they affect glaucoma? We shall see.

Although the ciliary body and the posterior surface of the iris are undoubted sources of the regenerated aqueous which flows through the eye; yet there is still another source, viz., the angle of the anterior chamber. This fact was first discovered two years ago by Ehrlich,* and subsequently confirmed by Ulrich of Strasburg, by a most ingenious method of investigation.

Ehrlich procured a living rabbit and injected it subcutaneously with a solution of fluorescin. (This substance is a brown-red pigment, highly soluble in serum, which in weak solutions fluoresces yellow or in front of a dark background, emerald green). He then immediately tapped the anterior chamber, and after the lapse of a few minutes, observed a shining green fluorescence which appeared to creep round the papillary margin of the iris from behind; simultaneously a slight yellow cloud was observed to start from the angle of the anterior chamber, and spread forward and blend with the previous current. This experiment has been confirmed by Ulrich, who used subcutaneous injections of ferrocyanide of potassium and subsequently injected a minute drop of perchloride of iron into the eye. This uniting with the ferrocyanide, immediately formed Prussian blue, and the entering streams were thus rendered visible. The same experiment was also tried in the rabbit's eye without the performance of paracentesis, and the result showed a marked flow from the angle, while but little or none appeared to come from the posterior chamber. This would seem to point out, that the angle plays a conspicuous part in furnishing the incoming current of aqueous, but we must bear in mind several facts which considerably detract from the value of the experiment. In the first place the course of the lymphatics in the rabbit's eye differs from that in man, for the structure of the parts around the angle is considerably altered; again, it is quite possible that the colouring matter could only pass through the tunica vasculosa with difficulty, and therefore appeared through the meshes of the ligamentum pectinatum around the angle, where the resistance was least. At present, therefore, we cannot recognise this as an undoubted source of lymph in the human eye.

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* *Deutsche med. Wochenschrift*, Nos. 2 and 3, 1882.

But allowing the truth of the above as regards the human eye, is the lymph which nourishes the retina, lens, and vitreous *identical* with the fluid normally in the anterior chamber? Is the fluid which has been observed to enter the anterior chamber by way of the angle, the same as that which is elaborated from the cells of the ciliary processes? I know of no facts which can be put forward as a distinct answer to these questions, and until they are settled, the complete elucidation of the problem must remain in doubt.

PART II.

So much light has been thrown upon the pathology of glaucoma during the last few years, that we are now able to speak on this point with precision.

In 1873 Leber* in a series of admirably conducted experiments showed that fluid injected into the anterior chamber passed under slight pressure through the ligamentum pectinatum into Schlemm's canal and the adjacent veins, and by the injection of coloured fluids proved that it was by osmosis, and not by direct filtration.

In 1876 Max Knies and Adolf Weber showed that in eyes blinded by glaucoma, the angle of the anterior chamber is almost invariably closed and impermeable.

In 1877 De Wecker established beyond doubt that the most important factor in primary glaucoma was a hinderance to the normal outflow, rather than an increased secretion.

In the same year Dr. Brailey from an examination of 53 cases not only confirmed the above results, but showed that in all cases the excretory channels were more or less occluded by either engorgement, or atrophy of the ciliary muscle and processes, and parts around the angle.

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* *Studien über den Flussigkeits wechsel im Auge,* Heidelberg Gesammte Augenheilkunde.

In 1878 Priestley Smith in his elaborate Jacksonian Essay gave very decided proof in support of the same doctrines, and showed moreover that if the pressure in the vitreous exceeded the aqueous by closure of the circumlental space, glaucoma was at once brought about.

Finally, in a series of papers in the *Royal London Ophthalmic Hospital Reports* for 1880, 1881 and 1882, Priestley Smith and Brailey added whatever was needed to set at rest any doubts on the matter. In the face of the above evidence it is no longer possible to doubt that this closure of the angle of the anterior chamber is the most fertile source of glaucoma, and that unless the escape can exactly neutralize the inflowing lymph, increased tension is the inevitable result. The question we have now to discuss is the manner in which this closure is brought about.

In the healthy eye a stream of fluid is constantly passing from the vitreous to the aqueous chamber through the circumlental space (*i.e.* the space which separates the margin of the lens from the ciliary processes). The ciliary processes are continually secreting fluid as above explained, into the vitreous chamber,* while the sole means of exit for the fluid lies at the angle of the anterior chamber, since no fluid can find its way through the posterior elastic lamina of Bowman into the cornea.* This is evident when we consider that the ciliary processes are continually secreting a supply of lymph into the vitreous chamber, while the sole exit for the waste lymph lies on the opposite side of the suspensory ligament, viz., in the angle of the anterior chamber. Now Leber has shown that the normal intraocular pressure is equal to about 25mm. or 1 *in.* of mercury, which though much lower than the blood pressure, is nevertheless intimately dependent on it. Anything therefore which raises or lowers the blood pressure, will proportionately raise or lower the tension in the eye, though from comparison with the case of other secretions, *e.g.* the salivary, it is probable that the amount is dependent, not only upon the dilation of the vessels, but is also under control of specific nerve fibres acting directly on the secreting cells. Moreover the intraocular pressure is subject in some degree to the influence of the fifth nerve and possibly of the sympathetic also. Under normal conditions the pressure of the aqueous differs but little from that in the vitreous. This, Priestley Smith, from a variety of experiments, has proved beyond doubt. Moreover he has shown that the suspensory ligament though possessing the same amount of intrinsic strength, cannot neutralize any appreciable excess of pressure without advancing from its normal position. By means of coloured injections in fresh human and pig's eyes, this observer has demonstrated that even so slight a difference of pressure as 2mm of Mercury between the two chambers (92mm in the vitreous and 90mm in the aqueous chamber), prevented the exit of fluid through the angle of the anterior chamber.

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* This lamina ceases about 1mm from the angle of the anterior chamber. Does not this appear to be a special provision of nature for allowing the ready exit of lymph at this point?

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* See Appendix note 5.

For the explanation of this we are indebted to Ad. Weber, who tells us that the periphery of the iris is driven forward against the cornea by the pressure of the ciliary processes from behind. For we see that the periphery of the iris is bowed forward in a sweeping curve, in the concavity of which the enlarged ciliary processes accurately fit. The apices of the ciliary processes being rounded posteriorly by the suspensory ligament, with which they are in contact, must if they swell, advance forwards and inwards, and in so doing carry the periphery of the iris before them, and thus close the angle of the anterior chamber.

Moreover, *post-mortem* examinations have shown me, that without a single exception, all the cases that I have either examined myself, or collected from the very complete statistics of Brailey, at Moorfields, present one or more of the following pathological characters, each of which would be enough to retard the escape of aqueous fluid, if not to stop the flow altogether:—

1. Atrophy of the ciliary processes, and contraction of the meshes of the ligamentum pectinatum.
2. Swelling and turgidity of the ciliary body and processes.
3. Contact or adhesion of the periphery of the iris (or the lens) with the periphery of the cornea.
4. Compression of the ligamentum pectinatum from pressure behind.

From an analysis of 88 specimens examined at Moorfields, I find:—

1. Angle of anterior chamber closed by contact of iris with the cornea	in 79 cases	
2. Excessive turgidity and inflammation of the ligamentum pectinatum and ciliary processes	,, 22 ,,	
Slight turgidity and inflammation	,, 32 ,,	Total 54
3. Excessive sclerosis and contraction of the ligamentum pectinatum and ciliary muscle and processes, with more or less atrophy	in 27 cases	
Slight sclerosis and contraction of the above	,, 42 ,,	Total 69

No atrophy at all in five cases. Of these, three were cases of serous iritis, in which the ciliary processes and irides were excessively swollen and inflamed; in the other two cases there was also considerable inflammation. The angle of the anterior chamber was wide open in five cases, and doubtful in four. Of these five, three were the above mentioned cases of serous iritis. In every case the ciliary muscle and processes were either inflamed or atrophied, while in thirty-one cases the blood-vessels of the ciliary region were enlarged.

Hence, I think we have before us sufficient evidence to establish the following propositions:—

1. Inflammation of the ciliary body, processes and pectinate region is present in a large number of cases of glaucoma.

2. In an equally large number of cases, more or less sclerosis and atrophy of these parts is to be found.

3. In a considerable number of cases both the above conditions exist together.

4. In every case one or other condition is present.

5. In every case, the immediate cause of the increased tension was traced to the retardation or arrest of the aqueous fluid; which was either due to the mechanical obstruction offered by the iris or lens, or (as in iritis serosa) to the violent inflammation and swelling of the parts around the canal of Schlemm.

We have now two questions to solve:—

1st. What is the cause of the apposition of the iris to the cornea? Is it due to the contraction dependent on the sclerosis and atrophy of the ciliary body and parts around the angle, or is it due to the pressure in the vitreous chamber being greater than that of the aqueous?

In a normal eye there is a complete partition between the aqueous and vitreous chambers, composed of the lens in its capsule, the suspensory ligaments, and the ciliary processes. Between the ciliary body and the lens is a narrow space—the circumlental space. Formerly the walls of the canal of Petit used to occupy this space and form the connecting link, but since the later anatomists have denied us this canal as being a postmortem myth,* we must content ourselves with the suspensory ligament as being the sole barrier between the vitreous and aqueous. This space is frequently very narrow, the margin of the lens being in actual contact with the more prominent ciliary processes.

It would at once suggest itself, that were either the lens or the ciliary processes to increase in diameter, the circumlental space would become almost obliterated, and the escape from the vitreous being retarded, the pressure in the latter would slightly exceed the aqueous. In consequence of this the lens and suspensory ligament would advance, and pushing the ciliary processes against the periphery of the iris, the latter would apply itself to the opposing cornea. Thus the exit from the aqueous is impeded and the current of the inflowing lymph being no longer equalised, the tension rises and glaucoma is established. Priestley Smith by injection into freshly excised eyes, established beyond doubt the fact that an exceedingly slight pressure in the vitreous above the aqueous, was sufficient to cause the above phenomena. Hence it becomes evident that we have here a ready solution to the problem. Priestley Smith sought it in the change in size which the lens undergoes, and brought forward a large number of facts, to show that the lens increasing in size occluded the circumlental space, and so induced the above described phenomena which cause glaucoma. There are, however, grave objections to this theory. Although the ciliary processes normally touch the margin of the lens, yet they do not form a flat curtain but a series of corrugated processes with grooved channels ·74mm in length and ·25mm in breadth. Hence in order to effectually block the way the lens would have to swell to an excessive size; but yet we lack pathological evidence that it increases in size at all. That the lens does normally increase slightly in size, up till about thirty years of age is well established, but Brailey has shown* in his elaborate investigation on the subject, that so far from the lenses of glaucomatous eyes being larger than normal, they are in many cases actually smaller.

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* *Vide* Appendix, Note 3.

We must therefore look for the obstruction in connection with the ciliary body and processes, and I can bring evidence to show that both the inflammation and swelling of the ciliary processes as well as their atrophy and consequent contraction are equally causes of glaucoma.

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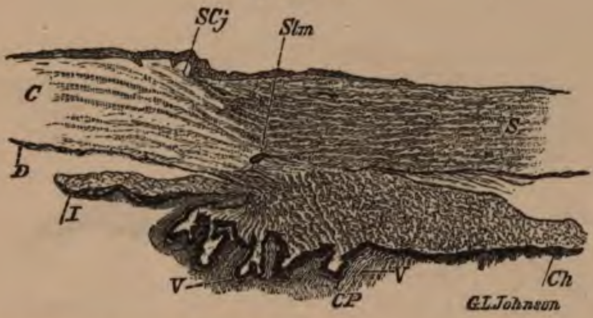
* Brailey. "On the Size of the Crystaline Lens in Glaucoma." *Royal London Ophthalmic Hospital Reports*, vol. x., part iii.,

Now a careful study of the ciliary processes will I think convince the reader, that if they become very congested and swollen, they will far more effectually block up the circumlental space than if the lens were to increase in diameter; for not only will they lap over and press against the lens, but the very turgidity of the ciliary processes will obliterate the channels which naturally exist between their folds, and thus complete the obstruction. Moreover this very swelling will tend greatly to impede the natural channels of escape through Schlemm's canal, and along the perichoroidal and sclerotic lymph channels. Frequently also, the inflammatory new formation becomes partly organized around the angle, causing adhesions which further prevent the escape of lymph. These adhesions are readily broken down, and differ essentially from the contracting cicatrices and adhesions which we find in the chronic forms of glaucoma.

That the ciliary processes do swell up, we have ample evidence, and although Brailey and others have raised the objection that such turgidity is never found in post-mortem examinations of glaucomatous eyes, I would urge that, in the first place after removal of the eye from the patient, this excessive hyperæmia and turgidity for the most part subsides, just as the post-mortem signs of œdema glottis or acute gastritis or or enteritis leave but little redness and swelling to

FIG. 2.



Section of the structures around the angle of the anterior chamber from an eye seized with acute glaucoma. Drawn from a microscopical specimen in the possession of the author, × 30.

SCj. Sclerocorneal junction. *Slm.* Canal of Schlemm. *S.* Sclerotic. *C.* Cornea. *Ch.* Choroid. *D.* Descemet's membrane. *I.* Iris. *V.* Vitreous. *CP.* Ciliary processes.

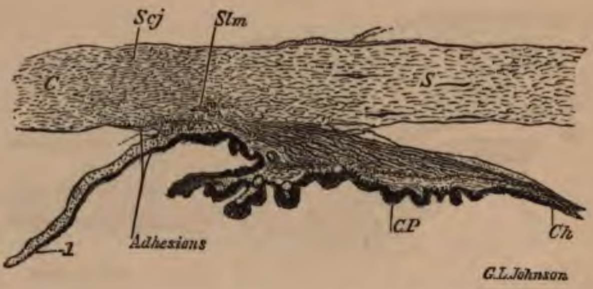
Notice the increased size of the ciliary body compared with that in fig. 3, also the great increase of nucleated cells.

account for the intensity of the symptoms. In the second place it is extremely uncommon for the surgeon to remove a glaucomatous eye until it has lapsed into the stage of glaucoma absolutum, when the acute symptoms would have long ago entirely subsided, and atrophy or sclerosis of the ciliary body have taken the place of the initiatory inflammation.[*]

[*] Notwithstanding the repeated denial that the iris and ciliary processes swell up as above described, I think I can prove beyond a shadow of a doubt that in many cases the turgidity does remain well marked after death, and the specimen of which Fig. 2 is a faithful copy, is a case in point.

That a contraction and atrophy of the ciliary body and processes is likewise a cause of glaucoma, we have strong reasons for believing. As we stated above, out of 88 post-mortem cases examined at Moorfields, nearly everyone showed distinct signs of sclerosis or atrophy of the ciliary processes and parts round about the angle of the anterior chamber and Schlemm's canal. Now all these parts are so intimately connected together that it is impossible to conceive the inflammation and swelling of any portion independent of the rest. Moreover the researches of Charcot and Duchenne go far to prove that sclerosis of the brain or spinal cord is always preceded by a previous inflammation; and the reseaches of Clifford Allbutt, Hulke, S. Mackenzie, Gowers, and many other Ophthalmologists go far to

FIG. 3.



Section of the structures around the angle of the anterior chamber in an eye, affected with chronic glaucoma, drawn from a specimen in the possession of the Author × 30.

Scj. Sclerocorneal junction. *Slm*. Canal of Schlemm. *S*. Sclerotic. *C*. Cornea. *Ch*. Choroid. *D*. Membrane of Descemet. *I*. Iris. *V*. Vitreous. *CP*. Ciliary processes.

prove that nearly every case of atrophy of the disc (white and grey atrophy of the optic nerve), is preceded by an optic neuritis or choked disc. Moreover this contraction and atrophy of the ciliary processes can be witnessed by the ophthalmologist as shown in the slow and gradual retraction of the ciliary processes and consequent recession of the lens.

Dr. Angelucci of Rome,* in a paper which has received less attention than it deserves, asserts that sclerosis of the minute arteries is usually at the bottom of the mischief in chronic glaucoma. He says "The arteries when sclerosed have their calibre diminished, and not only that but the length of the blood wave is also diminished. Thence results a venous stasis from a diminished systolic impulse, and also a failure of nutrition in the tissues supplied. Besides that, there is atrophy and obliteration of the lymphatic channels, which in the eye attains an extremely high degree; since as a result of vasculitis and perivasculitis the lymphatic spaces traversed by the blood vessels of the iris and choroid are obliterated. Venous stases are compensated by an increased secretion of lymph, but in glaucoma the lymph finding no means of exit, because the ways of filtration are closed, causes the augmentation of the intraocular contents, by which glaucoma is produced." Sclerosis of the arteries is undoubtedly common in chronic glaucoma, though I have failed to observe it in many cases. The idea is ingenious, and although it is open to grave objections, it is worth mentioning, as the influence of inflammatory changes in the minute veins in arresting the flow of lymph along the perivascular lymphatic canals, is apt to be overlooked.

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* "On the Etiology of Glaucoma." *International Congress*, 1881.

It is evident that the contraction and atrophy of the ciliary body and processes causes increased tension by their direct action just as the inflammatory condition does indirectly; by occluding or obstructing the outlet through the angle and Schlemm's canal, only in a somewhat different way; for whereas the former causes the obstruction by pressing the iris against the cornea, through the excess of pressure in the vitreous, the latter retards the escape of fluid by a continued, but ever increasing dragging of the base of the iris to the cornea, and thus occluding the angle. Moreover, by the contraction of new formed tissue, the lymph spaces and venous channels in the neighbourhood of Schlemm's canal, are drawn upon and ultimately obliterated. In serous iritis we have an exceptionally wide angle and it therefore forms an apparent exception to the above statement; but this exception disappears on examination, for although the angle is usually free and often very wide, yet serous iritis is the result of an inflammation of an exceptionally violent character of those very parts which are most concerned in the filtration process, and which therefore of all others lead to glaucoma when obstructed.

We will now conclude this part by the following propositions:—

1. The interior of the eye is nourished by fluid elaborated by the uveal cells of the ciliary processes and probably to a small extent by the pigment cells lining the posterior surface of the iris.

2. Under certain circumstances hitherto not correctly determined, aqueous fluid may enter the anterior chamber directly through the trabecular tissue at the angle.

3. All the fluid which does not enter the posterior aqueous chamber directly, circulates through the vitreous humour, and returning through the suspensory ligament by way of the circumlental space, enters the anterior chamber, where it joins with the stream from the posterior chamber.

The only possible normal means of escape for this fluid is by osmosis through the spaces of the ligamentum pectinatum (spaces of Fontana).

5. Within the spaces of Fontana, the current goes in three directions:—

(1). An anterior current which runs along the system of lymph canals, between the numerous layers of the cornea.

(2). A posterior current which circulates through the lymph canals of the sclerotic and communicates with the lymph channels of the choroid and retina, finally terminating in the ampulla of the sub-arachnoid space, (*vide* Frontispiece).

(3). The remaining portion passes into the canal of Schlemm, and according to some observers mixes with the blood in the anterior ciliary veins. (Schwalbe, Heisrath and others).

5. The lymphatics of the retina, choroid, cornea, sclera, and Tenon's capsule, all communicate with each other; partly through the lymph canals surrounding the venæ vorticosæ, which unite the perichoroidal and Tenonian lymph sacs, and partly through the communications between the subarachnoid, perichoroidal, and cribriform lymph spaces around the entrance of the optic nerve.

6. Under certain circumstances the pressure in the vitreous becomes greater than in the aqueous chamber; this causes the lens, suspensory ligament, and ciliary processes to advance in such a way, that the angle of the anterior chamber is compressed and the escape of lymph retarded. Inflammation and engorgement of the ciliary body and ciliary processes is the chief factor. The tension rises rapidly in proportion to the intensity of the inflammation, and acute glaucoma is the result.

7. Under other circumstances the inflammation of the ciliary region is so slow and insidious, as to fail to excite any acute symptom or marked increase of tension; but ultimately this inflammation passes into a stage of atrophy and sclerosis, and the contraction

of the new tissue reduces the size of the circular sinus, and lymph channels around the angle, and generally so binds the iris by adhesions to the cornea, that the outflow of lymph is unable to keep pace with the inflow, and chronic glaucoma is the result.

8. Lastly, the inflammatory process may be excessive, and the normal drains at the angle blocked up by particles of inflammatory lymph, and notwithstanding that the angle is open, and the pressure equal on both sides of the suspensory ligament, yet they fail to equalize the secretion. The tension rises and serous glaucomatous iritis, (acute serous glaucoma), is the result.

PART III.

We are now in a position to understand why iridectomy is so brilliant a success in acute glaucoma, why so useless for the chronic form.

We have noticed that in both acute serous glaucoma and in ordinary acute glaucoma, the ciliary processes ciliary body and iris were inflammed and engorged. We have a sudden inflammation and a sudden secretion poured into, perhaps an otherwise fairly healthy eye; we have intense pain with increasing blindness and limitation of the visual angle, together with all the other subjective phenomena of increased tension, but we have no chronic or long standing changes, no adhesions, no sclerosis. An iridectomy is beneficial in three ways. (1). It empties the anterior chamber and thus relieves the pressure from both chambers. (2). By removal of a portion of the engorged iris, it divides a number of vessels and thus allows a free drain of blood from the inflamed and overloaded vessels, not only of the iris, but also of the ciliary body and processes. By this means the latter are enabled to retract. The circumlental space is free, the lens and iris recede, and fluid can once more pass uninterruptedly from the vitreous to the aqueous. (3). Lastly, the section through the cornea allows of a more or less permanent drain which relieves the pent up spaces around the angle. If the inflammation subsides the normal angle of the chamber once more becomes patent, and the balance of the lymphatic circulation being restored, the tension falls to normal; and the passive hyperæmia set up in the vessels through their becoming squeezed, along their whole course, between the non-yielding sclera and the vitreous now becomes relieved and the vessels assume their normal calibre. Four and twenty years ago Von Graefe accidentally discovered this method of relieving the tension of acute glaucoma. At the time he remarked. "The theory is as yet infinitely darker than the empirical facts,"* and although the light of modern research has cleared up the theory yet we know of no method so certain or so successful as iridectomy for acute glaucoma.

IRIDECTOMY IN CHRONIC GLAUCOMA.

With *chronic glaucoma*, however, the case is different. Priestley Smith writes:†

"In chronic glaucoma, the simple non-irritative glaucoma of continental writers, as positive improvement of vision is seldom to be hoped for............the most that iridectomy can effect, is to remove the cause of the disease and thereby to arrest its further progress. Even this result, however, cannot be depended upon in every case; some surgeons go so far as to abandon the operation altogether in the purely chronic form of the disease."

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* *Memoirs, New Sydenham Society's Edition*, p. 357.

† *Jacksonian Prize Essay*, p. 201.

In 1863, J. Vose Solomon in a paper read before the Midland Medical Society,* remarked. "The statistics of the iridectomy operations are sufficiently favourable as to results on vision in acute primary attacks of glaucoma, but in the other class, subacute and chronic, are most unsatisfactory and discouraging."

Nor is the reason for these failures far to seek. Here the primary obstruction does not depend upon an acute inflammation, a hyperæmia, a vascular engorgement, or even upon a passive dilatation of the vessels, but upon slow and gradual changes in the ciliary body, and the tissues around the angle of the chamber, by which the connective tissue becomes increased, and contracting tends to narrow and imprison the lymph spaces, and to firmly tie down the iris against the contiguous cornea.

Again, the long continued pressure against the ciliary body and iris, caused in the first instance by an obstruction to the normal outflow, sets up structural changes which prevent them from returning to their natural position, and moreover, renders the iris so liable to tear, so rotten and inelastic, that to remove the iris up to its root, (an essental part of the operation for glaucoma), becomes an impossibility: hence the uncertain action of iridectomy in chronic glaucoma, the persistence of a slight excess of tension, the imperfect restoration of the anterior chamber and the recurrence of the malady after an interval of some months or years.

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* *Tension of the Eyeball,* by J. V. Solomon, 1865, p. 8.

Bearing these facts in mind and being aware of the unsuccessful results of iridectomy in chronic glaucoma; results which unfortunately I have had but too often to chronicle in the Royal Westminster Ophthalmic Reports, I examined the subject to see if some more rational method of treatment might not be adopted, or at any rate a method which, if not more successful in its results, would at least be unattended by so many objectionable features.

De Wecker writes: "I am convinced that with the progress of knowledge some other proceeding more simple and essentially more logical will be substituted."* That there are many objections are evident. In iridectomy there is a permanent loss of a portion of the iris, and therefore a permanent mutilation results. Again, the operation necessitates the employment of an anæsthetic and confinement to bed for a time, with the other accompaniments of an important operation. Moreover, the large size of the iridectomy, which is necessary to render it of any use, causes confusion of vision, from the dispersion of the rays of light which pass through the zonula.

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* De Wecker, *Ocular Therapeutics*, p. 270.

The chance of subsequent synechiæ, or of imprisment of a tag of iris in the wound setting up iritis or sympathetic ophthalmia, must not be overlooked, and the shock of so considerable an operation on a person in feeble health, frequently hastens the fellow eye into a similar condition of increased tension.

With the light that modern research has shed upon the subject, it appears to me that it is useless to seek for improvement by attacking the angle of the anterior chamber, since the destructive processes are in that region already too far advanced, while the relief afforded is insignificant when compared to the gravity of the operation. From other grounds, more especially from a study of the lymphatics, I felt convinced that the vitreous chamber offered a far greater chance of success to the operator. The spaces of Fontana had too long been occluded and the true channels of excretion too long obliterated by neoplastic tissue, the products of past inflammation to promise much hope of a successful result.

But, at least in an eye that is worth saving, the *choroid and sclerotic are generally healthy*,* and the lymph passages in relation to these parts patent for free communications between the vitreous cavity and the

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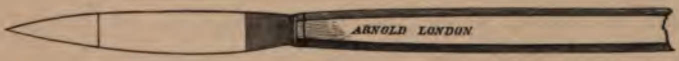
* In very few of the 88 cases of glaucoma examined by Brailey after removal, was the choroid found diseased. *Vide Royal London Ophthalmic Hospital Reports*, Vol. X. Part I. See also Priestley Smith's Jacksonian Essay and *Royal London Ophthalmic Hospital Reports*, Vol. X. Parts I and II.

Tenonian and perichoroidal spaces, and through them with the exterior. Moreover, as Brailey has pointed out, the appearance of little fluid cysts arranged round the pars ciliares retinæ, never occur in connection with normal tension, and appear to be an additional argument in favour of opening the vitreous cavity, and allowing its contents free communication with the lymphatic channels in its walls.

My method of performing the operation, which was originally suggested by Mr. Cowell and Mr. Rouse, is as follows:—

The patient lies on his back on the operating table with the head slightly raised on a pillow. The surgeon opens the lids with the stop speculum and fixing the eye with the forceps, turns it inwards so as to expose as much of the sclerotic to view as possible. He then with the other hand gently thrusts a double edged modified Wenzel's knife (see fig. 4.) through the sclerotic into the vitreous towards the centre of the globe. The point of the knife is entered about 4mm behind the sclerocorneal junction and should penetrate to the distance of about 1cm (nearly half an inch) the flat

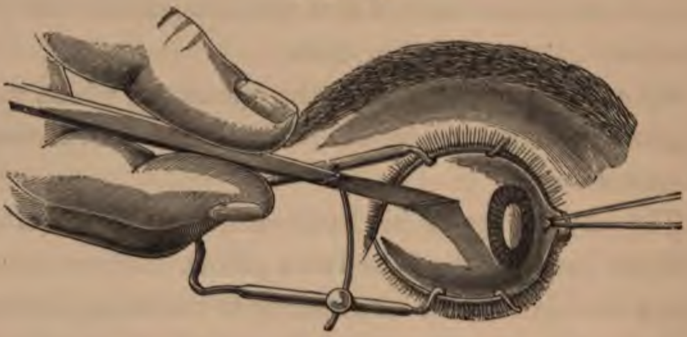
FIG. 4.



Knife used in paracentesis of the vitreous as made for me by Messrs. Arnold and Sons, actual size.

side of the instrument lying not quite parallel but somewhat oblique to the long axis of the eye. Any part of the globe will do, but it is better to avoid transfixing one of the muscles for obvious reasons.

FIG. 5.



The knife is then *very* slowly withdrawn, and if the tension is high, slightly turned on its axis so as to allow the lymph freely to escape. The speculum is then removed, and the eye bandaged up with a cotton wool pad for 24 hours, or longer if necessary, until the wound has begun to heal up. Unless the vitreous be *very* fluid, a condition which is nearly always accompanied by grave structural changes in the choroid and retina, and which therefore is unfavourable to vision, (even excluding the glaucomatous complication), the loss of vitreous need not be feared.* Lymph always escapes, but the pressure being on the inside of the globe (in this case a fortunate coincidence), as soon as sufficient lymph has escaped to allow the tension to sink below normal, the natural elasticity of the walls themselves close up the wound and prevent any further loss of fluid. It is necessary, however, to withdraw the knife with care, else this very elasticity which is of so much use in closing up the wound, will, acting like a ball syringe, tend to "squirt" out a portion of the more tenacious vitreous along with the lymphoid serum. No anæsthetic is needed, the pain of the increased tension being generally greater than that of the operation and the relief is always immediate and generally permanent.

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* See Mr. Hulkes' remarks on this point. Appendix, Note 1.

I have performed this operation in 6 cases myself and have seen it performed in about 10 more, while I have seen and taken notes of the after results of nearly as many other cases. In no case have I seen any bad results. My own cases and those of the surgeons at the Westminster Eye Hospital, who kindly performed the above operation several times at my request, are so satisfactory, that I feel the results, although few in number, fully bear out what my theory anticipated. In every case that I have seen has there been an instant reduction of tension, which has frequently never returned, or when it has, a second (or in two cases a third) repetition of the operation has reduced it permanently to normal. In two cases of chronic glaucoma the results were exceedingly encouraging. In the first case, the patient from barely distinguishing light from darkness, (V = shadows only, or $\frac{20}{nil}$), which was the case just previous to the operation on Dec. 28th, 1881, a few weeks after the operation could see $\frac{20}{30}$ well and $\frac{20}{20}$, *i.e.* full vision fairly.

In the second case (a woman, aged 42), a precisely similar result was obtained, the patient seeing V = $\frac{20}{20}$ fairly, or $\frac{20}{30}$ well, and reads J. 1½ at 16in., and when last seen 6 months after the operation no deterioration was detected. In several cases the sight has been improved; in all except one, the tension was permanently lowered, while in none did the sight deteriorate subsequently.[*] I may observe that this operation differs from any of the old operations, either Hancock's division of the ciliary muscle,[†] or Vose Solomon's "intraocular myotomy," quite as much as it does from De Wecker's or Quaglino's "subconjunctival sclerotomy," or Argyll Robertson's operation of trephining the sclerotic. In the face of these statistics the details of which I have here to hand, I hold that while iridectomy should be still selected as *the* operation *par excellence* for acute glaucoma, nevertheless in chronic glaucoma the statistics are so unfavourable that any alternative operation, which claims to do no more than arrest the progress of the disease, even if it only permanently relieves the pain deserves our serious consideration. The statistics of this operation, which for want of a better name, I will term Scleral Paracentesis, more than fulfil these conditions and therefore justify us in pressing its claims.

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[*] I have during the last few weeks received a letter from each of the above patients in answer to enquiries, and in each case the sight has remained as perfect as ever.

[†] See Appendix, Note 5.

APPENDIX.

NOTE 1.—To show that the operations of Hancock and Desmarres had nothing in common with the method I have described above, I need only quote an extract from a very able paper read before the Royal Medical Chirurgical Society by Mr. Hulke, as reported in the *Lancet*. "To avoid these alleged disadvantages, paracentesis scleroticæ has been advocated by Middlemore, Desmarres, and Hancock, as a substitute for iridectomy. Middlemore proposes *to evacuate the turbid diffluent vitreous humour* with a grooved needle, and to replace it with a syringeful of clear water. But except in very old cases" Mr. Hulke truly remarks "the vitreous humour is much too firm to flow out along a grooved needle, and probably few English surgeons would adopt Desmarres' suggestion of introducing a probe and breaking it up. Mr. Hancock, considering a spasm of the ciliary muscle to be an essential part of glaucoma, divides this muscle by striking a knife through the ciliary region. But Mr. Hulke has demonstrated by microscopical examination advanced atrophy of this muscle in many glaucomatous eyeballs; whence it follows that the ciliary muscle is not actively concerned in maintaining the glaucomatous process. In all probability, the success of Mr. Hancock's operation, is solely due to the draining away of some of the superabundant fluid." *Lancet*, 1860, vol. ii, p. 62.

NOTE 2.—It is astonishing that so careful an observer as Dr. Brailey should have denied the existence of spaces in the vitreous, but that there are natural lymph channels in the vitreous, *i.e.* channels which afford a more ready passage to fluids than other parts of the vitreous, is supported by a whole host of evidence. Klein says on this point:—"The substance of the vitreous humour in man and mammals appears, according to almost all observers (Brücke, Hannover, Bowman, Doncan, Stilling, Iwanoff, Gerlach, Smith, Schwalbe, and many others), arranged in layers separated from one another by clefts. These clefts possess in the peripheral parts, a more concentric arrangement, and they radiate from here towards the centre." *Atlas of Histology*, p. 358. For those who are interested in this subject, I would recommend the perusal of Dr. D. Smith's admirable paper on the composition of the vitreous, (*Lancet*, 1868, vol. ii, p. 376); also Stilling's article "Eine Studie über den Bau des Glaskörpers," *Archiv. für Anatomie*, Band xv, Heft 4. But the reader can easily assure himself of their existence by a simple experiment, which I have several times performed, of injecting the vitreous of a bullock's eye with magenta or aniline blue, freezing in gum and then making a section through the vitreous. Hardening the eye for some weeks in chromic acid solution, is said by many observers to answer and the result is pictured in *Quain's Anatomy*, vol. ii., fig. 359. I have repeated it several times, but I confess I have never succeeded in demonstrating the spaces by this method.

NOTE 3.—*Tenon's Capsule.* This remarkable fascia which plays so important a part in the lymphatic system of the eye, has strange to say, been almost overlooked by anatomists. Many describe it as being a capsule investing the eye and merely serving the purpose of allowing the eye to roll freely in response to the muscles. Of all writers on anatomy Richet appears to be the only one who has done justice to the subject, and who has conceived the importance of recognising its continuity with the dura mater and its cavity, "Tenon's space," as being a direct prolongation of the arachnoid cavity of the brain. See Richet, *Traité Practique d'Anatomie*, Lauwereyns, Paris, 1877; and in English, Macnamara's *Manual of the Diseases of the Eye*, Churchill, 1882.

NOTE 4.—*The canal of Petit a postmortem myth.* Stricker in his *Handbook of Histology*, vol. iii, p. 345, says:—"My own researches support the view entertained by Henle as in frozen eyes at least, I was unable to discover any ice in the canal." My own view is that the canal of Petit is a well marked space in the eye after death and can readily be inflated with air, but non-existent in the living eye; the space being merely due to the shrinking of the vitreous at this point. The whole question, however, is by no means a settled one. Even the very existence of the hyaloid membrane has been denied by many of the first anatomists of the day, (*e.g.* Henle, Schwalbe, Iwanoff).

NOTE 5.—Hancock in describing his operation for glaucoma, writes thus:—"The operations of Middlemore and Desmarres were introduced for the avowed purpose of relieving intraocular tension, by the evacuation of fluid. My operation, on the contrary, is introduced for the avowed purpose of relieving the constriction of the several coats of the eye, by division of the ciliary muscle." *Lancet*, 1860, vol. ii, p. 338. With regard to the spasm of the ciliary muscle no comment is needed. It has long been shown to be a theory which had no facts to support it, and indeed Hancock himself had to look for support in the direction of his colleague Mr. Hogg, who stated that in looking through the tube of the microscope, he observed in two cases the ciliary muscle to have been "hypertrophied and anything but in a state of atrophy," which latter Hulke and Bowman had pointed out to be a fatal objection to the "spasm theory."

These ideas could not now exist for a moment, in the light of modern research.

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